blood corpuscles and inflammation of the neuroglia follows, leading secondarily to destruction of the nerve elements. General paralysis, then, may be designated as *a diffuse interstitial encephalitis, terminating in brain atrophy.*

Zur pathologischen Anatomie der Dementia paralytica. LUDWIG MEYER. Neurologisches Centralbl., 1890, No. 20, p. 610.

This is a criticism of certain points in the preceding article by Mendel, citing the conclusions that general paralysis is a diffuse interstitial encephalitis, and the conclusions on the microscopic examination of the brain of the dogs. Meyer claims that essentially the same findings have been shown by him to exist in a considerable number of cases, and since Mendel casts doubt on the significance of these observations, either through doubt as to the diagnosis or because of the small number of cases, Meyer reviews his own contributions to the subject of *dementia paralytica* extending over a number of years. In 1858 he claims to have advanced proof that in typical cases the disease takes its course in febrile exacerbations, and may therefore be classed with the chronic febrile diseases as a meningo-encephalitis. The anatomical proof of this was published in the *Centralbl. für med. Wiss.*, 1867, Nos. 8 and 9. The accumulation of the nuclei and cells in the walls of vessels was described, and the change of the ganglion cells in atrophic brains was described as a result of vascular degeneration. Meyer agrees with Mendel that the question of the primary changes can only be settled by a study of those cases with a very rapid development; but this rapidity must without doubt be looked on suspiciously, for since "the diagnosis must be absolutely certain" it is necessary that the time of development as well as of the existence of the typical symptoms should not be too short. Meyer claims that with the limitations just mentioned he has given the essential anatomical changes of the disease in his work on the Pathological Anatomy of Dementia Paralytica (*Virchow's Archiv*, 1873, pp. 270–303.) As far back as then he said "only those changes in the brain can be looked on as pathological which appear and are constantly observed with the first distinct symptoms of disease," and "cases of very short course must serve exclusively as the basis for investigation." "Brains with appearances of atrophy must be excluded, or must be admitted only with great reservation." Meyer found 20 cases answering these conditions; and among other things, the normal brain weights went to indicate that the cases fulfilled the required conditions; and these were further strengthened by some of the phenomena of the course which resembled a severe meningitis, or there were headaches in the beginning, maniacal outbreaks resembling febile delirium, convulsions, paralytic attacks, etc. In all the cases there were early autopsies. There was cell-proliferation of the vessel walls. Meyer's conclusion at that time was that, apart from the chronic meningitis which was not always present, the changes at the beginning of the disease were confined to these vascular changes, which were claimed to be inflammatory. Examination of the substance of the brain gave an entirely negative result.

MANNER OF DEATH.

De la mort dans la paralysie générale. JEAN L. BARAZER. Thèse de Paris, 1890, No. 179.

Barazer considers that the question of the mode of death in general paralysis may be reduced to simpler proportions than in Jamin's thesis of 1887. If it is true that the causes of death may be innumerable, it is also incontestable that the patients always die, or at least almost

always, in the same manner, and that all the cases of death, with almost no exceptions, may come under the general primary disease.

The forms of death are as follows:

a. By the direct manner. { Apoplectiform attacks. / Epileptiform " / Maniacal excitement. / Vices of nutrition and trophic troubles { Paralytic / Marasmus and Bed-sores.

b. By the indirect manner. } But intimately related to

c. By accidental causes. } the cerebral lesion.

The natural form of death is by marasmus or paralytic wasting. This is the natural termination of the disease and also the most frequent, other forms of death being only complications. Christian and Ritti found this manner of death in 52 out of 119 men, and in 9 out of 20 women. The author adopts Charcot's theory of the central origin of *decubitus acutus*.

COMPLICATIONS.

Contribution à l'étude du mal perforant dans la paralysie générale. HILDEVERT BERTHÉLEMY. Thèse de Paris, 1890, No. 126.

The author considers historically the question of the origin of perforating ulcer of the foot, and of the three theories as to its origin, the mechanical, the vascular, and the nervous theories, he favors the latter, claiming that the nervous lesion is always present, while the vascular changes may be absent. Perforating ulcer is usually considered a rare complication in general paralysis, but Berthélemy claims that it is not so rare as is supposed, and that published observations are so few because attention has not been sufficiently called to the condition, and he is able to present eighteen observations, all from French sources, four being personal. In two cases the patients had suffered from a preceding locomotor ataxia, and the combined action of the tabes and the general paralysis producing the ulcer is admitted, while in two other cases alcoholism was doubtless a similar causal factor, but in the remaining fourteen there was no complication, and the general paralysis was looked on as the direct cause of the ulcer. A striking fact brought out in the study of the cases was that in eight of the eighteen cases it was noticed that under the evident influence of the ulcer undoubted remission occurred in the course of the general paralysis. The same fact was noted by Christian in publishing his observations on *mal perforant* in 1882, and he claimed that almost all remissions in general paralysis came as a sequel of prolonged suppurations. Berthélemy suggests that such remissions following suppurations give a basis for the therapeutic employment of revulsive measures such as setons in the neck or the actual cautery. The author reaches the same general conclusions as Marandon de Montyel in his thesis of 1888:—1. Progressive general paralysis is as justly a cause of perforating ulcer as other diseases of the nervous system. 2. The infrequency of published observations on perforating ulcer point to the fact that the complication should be carefully looked for to be discovered, and that attention has not been sufficiently called to it. 3. Perforating ulcer shows itself by preference in those general paralytics who have been alcoholics, or who have at least had some excess in drinking. 4. Perforating ulcer, judging by those cases which we have at command at present, favors remissions in general paralysis in those paralytics who have it.